

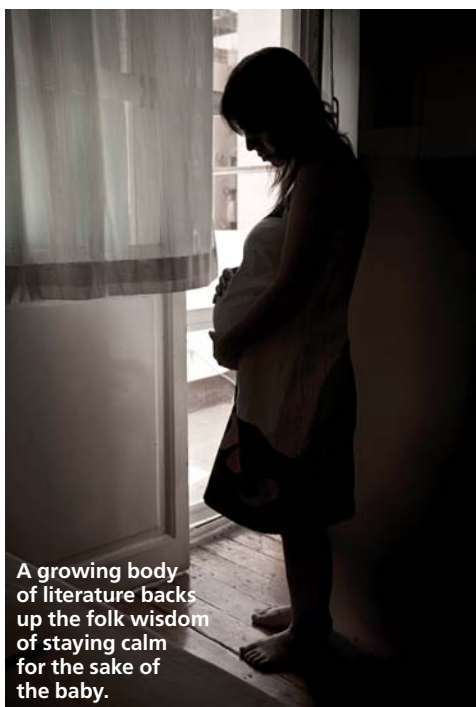
## An Emotional Bond

### The Relationship between Maternal Stress and Offspring Disease

Studies have suggested a potential association between maternal stress during pregnancy and an increased risk of specific diseases in offspring, prompting calls for a closer examination of stress as an environmental health factor. A new comprehensive study based on a large population-based cohort in Denmark explores this association further by examining maternal stress during pregnancy and the development of a wide spectrum of pediatric diseases [*EHP* 119(11):1647–1652; Tegethoff et al.].

The researchers examined prospective data for 66,203 mother–child pairs enrolled in the Danish National Birth Cohort. The mothers gave birth between 1996 and 2003, and the median child age at the end of followup was 6.2 years.

The researchers assessed two types of stress during each woman's pregnancy—"life stress" and "emotional stress"—based on information reported by mothers at about 30 weeks' gestation. Life stress refers to the mother's perceived life burdens, e.g., work, housing, and human relations. Emotional stress refers to the mother's feelings, e.g., irrational fear, hopelessness, and irritation. The researchers also collected data on disease diagnoses reported to the Danish National Hospital Register for the study children.



A growing body of literature backs up the folk wisdom of staying calm for the sake of the baby.

On average, mothers reported both life and emotional stress as being stronger before birth than after. The researchers observed associations between maternal life stress during pregnancy and a variety of childhood ailments during followup, including infectious and parasitic diseases, mental and behavioral disorders, and diseases of the eye, ear, skin, respiratory system, digestive system, musculoskeletal system, and genitourinary system. Maternal emotional stress during pregnancy was associated with an increased risk of infectious

disease only—a finding that contradicts some previous studies. The authors write that the discrepancies may result from differences across studies in classifying emotional stress or disease outcomes.

Limitations to the study include a lack of data on the timing of mothers' stress during pregnancy (each organ system has a specific critical window of intra-uterine susceptibility) and the possibility of uncontrolled confounding factors such as chemical exposures. However, the findings overall corroborate and build upon those of earlier studies and underscore the need for strategies to reduce maternal stress during pregnancy. They write that future studies should look in more detail at offspring diseases related to maternal stress and should focus on determining the underlying mechanisms, which may help improve preventive approaches and interventions.

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## Photochemical Finish

### Assessing the Genomic Impact of Secondary Air Pollutants

Most studies and regulations of air pollutants address individual substances in their unaltered primary form. But people typically are exposed simultaneously to multiple pollutants, and primary pollutants can be photochemically transformed in the atmosphere into secondary, tertiary, and additional generations of substances. Scientific limitations have hampered study of these real-world conditions and impaired our understanding of exactly how air pollutants affect health. Findings by a team of University of North Carolina researchers provide insight into the biological effects of air pollution and suggest that current air pollution regulations and mitigation approaches should consider products of atmospheric reactions, which have received scant attention in the past [*EHP* 119(11):1583–1589; Rager et al.].

The researchers used a toxicogenomics approach to evaluate the impacts of primary and secondary pollutants on human epithelial lung cells. They found that 4 hours of exposure to a mixture composed largely of primary pollutants (nitric oxide, nitrogen dioxide, and 55 hydrocarbons) at environmentally relevant concentrations changed the expression of 19 genes, compared with 709 altered genes following similar exposure to a mixture of primary and secondary pollutants (including ozone, formaldehyde, and peroxyacetyl nitrate). They also found that release of the enzyme

lactate dehydrogenase, a marker of cell injury and death, was 9 times higher after exposure to the secondary pollutants compared with the primary pollutants.

The authors identified molecular networks related to sets of genes expressed in response to exposure. One network was identified for genes affected by the primary pollutant mixture, which included proteins relevant to cancer biology. Twenty-five networks were identified for genes affected by the secondary pollutants, including those relevant to biologic processes associated with cancer; cellular movement, growth, and proliferation; tissue development; and cardiovascular disease. The authors also identified transcription factors that might regulate responses to the pollutant exposures. Both primary and secondary pollutants affected hepatocyte nuclear factor 4 $\alpha$  signaling, which plays a role in the function of organs such as the kidney, liver, and intestine.

The authors acknowledge this study offers only a glimpse of what's occurring in real-world conditions. Future studies will need to consider long-term exposures, defense mechanisms in living beings, specific health impacts linked with altered gene expression, many other primary pollutants, and myriad combinations of primary, secondary, and subsequent generations of pollutants. In addition, research is needed to pin down the roles of individual substances within any given mixture. But these initial results provide insights into what is missing in current knowledge and should spark more in-depth investigations.

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